ASTHMA,

WITH AN ANALYSIS OF EIGHTY CASES, WITH ESPECIAL REFERENCE TO ITS RELATION TO LOCAL DISEASES OF THE UPPER AIR TRACT.

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In reviewing the literature of asthma, at the present day, when our knowledge of this disease has become systematized and definite, one is particularly struck with the exceedingly vague and indefinite views which have prevailed in regard to it up to comparatively recent times, and particularly with the curious theories which have been advanced to account for the symptoms which characterize it; for, while ancient observers could not fail to have their attention prominently attracted to it by the peculiar character of its manifestations, yet I find that it is rather as a symptom than as a disease, that most writers deal with it. Even as late as 1874, we find Bennett[1] devoting only a few lines to its consideration as a symptom of emphysema and bronchitis, rather than as a distinct disease, while Watson, in his classical work on the *Practice of Physic*, although devoting a chapter to the subject, makes the somewhat *naive* confession that he has never listened to a case by auscultation. As early, however, as 1852, we find careful observers searching for some rational explanation of the peculiar symptoms which characterize this curious affection. As far as we know, Bergson[2] was the first to make it a distinct disease, although its individuality was denied by Rostan[3] and by Beau.[4]

The ancients believed the disease to be due to spasmodic contraction of the bronchial tubes. This view was, however, controverted by Laennec, who cites, as an argument, those cases of asthma in which we have puerile breathing over the entire chest, thus showing that the capacity of the lungs may be increased during a paroxysm rather than diminished. Copeland calls the same cases nervous asthma, while Walshe suggests the term hæmic asthma. Dr. Bree takes a different view, believing the disease due to some specific irritant in the air tubes and that the asthmatic paroxysm is an effort to expel these so-called irritating humors.

Beau,[5] whose treatise has already been referred to, believes that all cases develop from a primary bronchitis.

Todd[6] regards the disease as humoral, comparing it to gout or rheumatism, and believes the *materies morbi* affects the respiratory centre; while Budd[1] divides it into two forms; one depending upon cardiac disease, emphysema, etc., the second form due to a spasm of the respiratory muscles. The mere fact that an attack of asthma is always preceded by a feeling of a want of air and increased respiratory effort is enough to controvert this view. That this view has been held, though, for some time, is shown by the fact that so recent an author as Wintrich[2] advances essentially the same idea.

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[1] Principles and Practice of Medicine. American edition. Philadelphia, 1874.

[2] Recherches sur l'asthme, 1852.

[3] De l'asthme; Gaz. des hopitaux, No. 31, 1856.

[4] Traité Clinique et pratique de l'auscultation. Paris, 1856.

[5] Arch. Générale, vol. 78, p. 155.

[6] Medical Gazette, 1850.

The first to write a really exhaustive work on asthma was Henry Hyde Salter.[3] His work has become a standard one, and his views have been adopted by most subsequent writers on general medicine. He makes the following propositions:

First. Asthma is essentially, perhaps with the exception of a single class of cases, a nervous disease, the nerve centres being the seat of the essential pathological condition. *Second.* The phenomena of asthma, distressing sensation and demand for extraordinary respiratory efforts, immediately depend upon spasmodic contraction of the cells of unstriped muscular fibre in the bronchial tubes. *Third.* The phenomena are excito-motor or reflex actions. *Fourth.* The extent to which the nervous system is involved differs much in different cases, being, in some, restricted to the nervous apparatus of the air-passages themselves. *Fifth.* In a large number of cases, the pneumogastric, both gastric and pulmonary portions, is the seat of the disease. *Sixth.* In a large class of cases the nervous circuit involves other nerves beside the pneumogastric. *Seventh.* There is still another class of cases in which the irritation is central. *Eighth.* In a certain proportion of cases the irritation is humoral.

We find here that the bronchial spasm-theory of the ancients was fully adopted as the result of large clinical observation and study, and maintained by Salter in the several editions of his popular work. This theory is the one adopted by Biermer.[4]

We see, then, that, according to Salter, asthma is essentially a neurotic disease, and this theory, with some modifications, is the one adopted at the present day. Dr. Burney Yeo[5] attempts to draw an analogy between the extreme distention of the lungs in asthma and abdominal distention in hysteria. This observation is curious perhaps, but scarcely harmonizes with clinical observation. Morton[6] draws a similar analogy between asthma and spasmodic croup. The sudden nightly attacks, with daily remissions; a certain periodicity observed in the attacks of both diseases; a dry first stage and moist second stage; all seem to him to point to a certain similarity between the two diseases. Another curious observation which he makes is that the tendency to asthma begins at about the time when the tendency to spasmodic croup ceases. He believes that both croup and asthma are due to disorders of innervation of the larynx, on the one hand, and of the bronchial tubes, on the other, the immediate cause of the paroxysm being excess of venous blood in the medulla. This observation is also incorrect by the decided error in both premises.

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[1] Med. Chir. Transactions, vol. 23. London, 1840.

[2] Virchow's Handbuch der Path. und Ther., Bd. v. Ab. 1.

[3] On Asthma; Its Pathology and Treatment. London, 1860.

[4] Ueber bronchial Asthma, Sammlung klinische Vorträge, 1875.

[5] London Practitioner, 1881.

[6] British Medical Journal, January 22, 1877.

In the diligent research after the hidden causes of disease so characteristic of the present day, Leyden[1] claims to have discovered certain elements in the sputa of asthmatics, known as "Leyden's crystals," and misnamed, for some reason, Charcot's crystals.

Ungar, of Bonn, in an investigation of thirty-nine cases of spasmodic asthma, as recently as 1882, found these crystals in the sputa, but also found that they increased in number the longer the sputa stood, thus, to a certain extent, vitiating Leyden's theory.

Quite recently, Dr. Pfuhl[2] relates the case of a soldier whose sputa contained large numbers of the crystals of Leyden, without any evidence of asthma. Further, he has examined the sputa in 855 cases of pulmonary disease, and has only found the crystals in the one case mentioned above. An attack of asthma, as we know, consists in the occurrence of more or less well marked symptoms of oppression of breathing, with a certain amount of periodicity, coming on suddenly, generally at night, and lasting for several hours, the dyspnœa obtaining both during inspiration and expiration, while the cessation of the attack is accompanied by a more or less profuse serous and sero-mucous expectoration. This series of phenomena has been explained by the writers quoted above on the theory that this dyspnœa is due to the contraction of certain muscular fibres, which anatomists have demonstrated as existing in the bronchial tubes, down to their smallest ramifications. It is difficult to understand why the early writers did not vigorously question this conclusion as failing to explain rationally the phenomena of a paroxysm of asthma. The only explanation of this is that their knowledge of pathological processes was unequal to supplying a more plausible theory.

In 1872, however, we find the spasm-theory called in question, and what, to my mind, is a far more plausible one advanced by Weber,[3] who was the first to teach us that the cause of the paroxysm lay in a paresis of the vasomotor nerves presiding over the vessels of the bronchial mucous membrane. Under the influence of this vasomotor paralysis,

[1] Virchow's Arch., Bd. 54, 1871.

[2] Deutsche med. Zeitung, 1887, No. 76.

[3] Tageblatt der 45te Naturversammlung zu Leipsig, page 159, 1872.

there occurs, from some cause, a sudden letting up of the control which is exercised over the calibre of the bloodvessels, whereby they become distended to such an extent as markedly to interfere with the passage of air through the bronchial tubes. This paralytic condition having lasted several hours, the membrane maintaining a dry condition, as is always the case in the first stage of the inflammatory processes, there follows an escape of serum and sero-mucus, thus relieving the engorged bloodvessels, which soon regain their normal calibre, coincident with the cessation of the paroxysm. We thus have a thoroughly rational and plausible theory in explanation of the symptoms of spasmodic asthma. As to the causes, however, of the disease, little has been said further than the causes already stated, as laid down by Salter. Weber's paper, however, was followed by a series of clinical observations which largely lent weight to his theory and also threw much light on the causes of the disease. The first observation of note in this connection was that of Voltolini,[1] who reported a case of asthma, due to the existence of nasal polypi, as shown by the fact that the asthma promptly disappeared on the removal of the nasal growths. This observation was followed by a large number of similar reports by Hanisch, Porter, Daly, Todd, Spencer and others, as noted by Mackenzie,[2] and gave rise to voluminous discussions by Shafer, Fränkel Bresgen, Hack and others, not only on asthma as a reflex disease due to nasal polypi, but also as due to other nasal disorders.

As before stated, the literature of this subject has assumed large and voluminous proportions, but it still inclines itself to the subject of asthma as a reflex disease. Now, I do not propose to enter into this subject of reflexes, which has always seemed to me as a term oftentimes used as a cloak to conceal our ignorance of the direct relation between cause and effect, but I am convinced that, in very many instances of morbid symptoms occurring as a result of reflex disturbance, we can offer a more rational explanation than "reflex," in the sense in which the term reflex is so often used at the present day. Following Voltolini's observations that nasal polypus was the cause of asthma, and intimately connected with the same line of investigation, came the study of hay-fever. The first impetus, as I think, to this line of investigation, was a paper by Daly.[3]

Up to this date, hay-fever had been regarded as simply a periodical coryza or influenza, in which the paroxysms were characterized by the same symptoms as are met with in an ordinary cold in the head. As a matter of fact, however, acute rhinitis and an attack of hay-fever differ in a marked way, in many respects. This fact was soon recognized, and a new name was given to hay-fever, *vasomotor rhinitis.* We have, thus, suggested a connection between the two diseases, and, as a clinical fact, the two diseases are intimately connected; for, as we know, a large number of hay-fever patients suffer from asthma, following soon after the onset of their nasal symptoms. A natural division of cases of asthma into *hay-asthma* and *perennial asthma* is thus made, the one term being applied to those cases that are attended with hay-fever, the other to those cases in which asthma occurs without reference to seasons and without the preceding influenza. The question now arises, What, if any, connection exists between the two, or how far are these two diseases one and the same; and, again, what is the connection between hay-fever and asthma, and are they not, in many respects, the same disease? In a paper read before the American Climatological Association, May 28,[1] 1885, I first advanced the view that hay-fever and perennial asthma are virtually one and the same disease, the one being a vasomotor rhinitis, the other being a vasomotor bronchitis, the paroxysms being excited, in each case, by some peculiar atmospheric condition. The atmospheric condition, as we know in hay-fever, is the presence of the pollen of flowering plants or some other vegetable emanation; whereas the atmospheric condition in perennial asthma, as we may designate those cases of asthma which occur during the whole year and do not depend upon hay-fever, is dependent upon some obscure element which we are, as yet, unable to trace with the same degree of definiteness as we are enabled to trace it in hay-fever. Hay-fever is dependent upon three conditions:

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[1] Die Anwendung d. Galvanokaustik. Wien, 1872, p. 246, 4th ed.

[2] Diseases of the Throat and Nose. American edition, vol. ii. Philadelphia, 1881, p. 357.

[3] Archives of Laryngology, vol. iii. p. 157.

First. A neurotic habit, as was conclusively shown by Beard.[2]

Second. The presence of pollen in the atmosphere, as shown by the unrivalled experiment of Blackley.[3]

Third. A disordered condition of the nasal passages, as shown by Daly.[4]

Now, the view that I advocated is that asthma also is dependent on three conditions:

First. A general neurotic condition, as demonstrated by Salter.[5]

Second. A diseased condition of the nasal mucous membrane (not the bronchial).

Third. Some obscure condition of the atmosphere exciting the paroxysms.

The view as regards the neurotic condition is generally accepted; that as regards the atmospheric condition, I think, is one which must be generally accepted by all observers when we consider the diurnal and seasonable periodicity of the paroxysms. As regards the nasal condition as a predisposing cause of the attacks, the view is a novel one, and, naturally, would be looked upon as the hobby of a specialist. In my original paper, I made this assertion, that "a large majority, if not all, cases of asthma were dependent upon some obstructive lesion in the nasal cavity. This is evidenced by the immediate relief from the exacerbation by the use of cocaine in the nose in every case in which I have tried it, and, furthermore, by the cure of so many cases by the removal of the obstructive lesion in the upper air-passages."

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[1] New York Medical Journal, April 24, May 1, 1886.

[2] Hay-fever or Summer-catarrh. New York, 1876.

[3] Hay-fever. London, 1873.

[4] Loc. cit.

[5] Op. cit.

This paper was read two years ago. The views there stated I would repeat with even more emphasis, for subsequent clinical observation has only served to confirm me in my belief of their correctness. That the lesion in a paroxysm of asthma is a vasomotor paresis of the bloodvessels supplying the bronchial mucous membrane, and not a bronchial spasm, I do not discuss, but accepting this theory as the only one which can explain the symptoms, the question arises, What is the connection between the nasal mucous membrane and that of the bronchial tubes? I have already written[1] fully on the subject of the great respiratory functions of the nasal mucous membrane, and I need not repeat them here at length. In brief, the most intricate, the most delicate and most important part of the whole respiratory tract lies in the nose, in that mass of bloodvessels which we call the turbinated tissues, and which serve to supply the inspired air with moisture, by pouring out upon the surface of the mucous membrane a large amount of water—sixteen ounces in the course of the day—by which the inspired air becomes saturated with moisture, this function being necessarily regulated with an extreme degree of nicety of adjustment. This establishes, in what way or through what nerves or ganglia I do not discuss, but to my mind does unquestionably establish a most intimate connection between the two portions of the respiratory tract. The blood supply in the nose being regulated by the same vasomotor tract as that which regulates the blood supply of the bronchial tubes, a disturbance in one region is liable to be followed by a disturbance of the other; a morbid condition in one region renders the other especially susceptible to diseased processes. This, briefly, is the history of the connection between the two parts. Hence, we see, therefore, how a diseased condition in the nasal cavity may predispose a neurotic patient, under favorable atmospheric conditions, to an attack of asthma; the same line of reasoning, as will be noted, being followed here as in the case of hay-fever. This connection between the two regions I have not found alluded to by any writers, and yet I cannot but think that Hyde Salter[2] must have entertained a somewhat similar idea when he says, in speaking of the causation of asthma, that we may divide the cases into two classes: First, cases in which the essential cause of disease, "that which constitutes the individual an asthmatic," is some organic lesion, possibly not appreciable, either in the bronchial tubes or some part physiologically connected with the bronchial tubes. Second, cases in which the organic lesion does not exist, in which the tendency to asthma is due to something from within, not from without, in which the cause of disease is a congenital and possibly inherited idiosyncrasy. The large clinical observation and study which were the basis of Salter's classical work, could not fail to have impressed upon him that a diseased condition of the upper air-passages was prominently active among the predisposing causes of asthma. It would seem a rather broad statement to make that all cases of asthma find their predisposing cause in intranasal disease, and yet I am very confident that it is very largely, if not entirely, true. Certainly, in my own observation, I have seen no case in which this could not be stated. The question suggested by Mackenzie[1] here arises, What constitutes a typically healthy nose? Mackenzie seems to think that there is a very large difference in individuals, even in health, and rather suggests that a typically healthy nasal cavity is difficult to find. On the contrary, I think that every nasal cavity which shows a departure from the normal type should be regarded as in a diseased condition. The true test, however, in these cases is this: if we find diseased conditions, the removal of which cures an asthma, my proposition, in that individual case, is certainly established. I make the general statement that all cases of asthma have intranasal disease, without giving definite proportions. This may seem rather broad, when we find eminent physicians of the present day, such as Fothergill, Flint, Loomis and others, adhering to the old theory of bronchial spasm, and not mentioning pathological conditions in the nasal chamber as a possible cause of the disease. That my view is by no means entertained by laryngologists is shown by the fact that Böcker[2] makes the statement that asthma is seldom associated with polypi and seldom cured by their removal, and that hay-asthma is caused by direct irritation of the bronchi, and, further, that, normally, asthma cannot be produced in the nose.

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[1] Loc. cit.

[2] Op. cit., page 61.

This first assertion of Böcker seems, to me, to be a very grave reflection on his skill as an operator. In the *Union for Internal Medicine*, May and July, 1886, Lublinski, Heyman, Böcker and Krause assert that, in many cases, asthma is independent of a pathological condition of the nasal passages. Schech,[3] however, states that, in sixty-four per cent. of cases of asthma, he found intranasal disease, and further adds that there must be associated excessive nervous irritability—in other words, the neurotic habit.

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[1] Hay-fever, London, 1885, page 25.

[2] Deutsche medicinische Wochenschrift, 1886, Nos. 26 and 27.

[3] Münchener medicinische Wochenschrift, 1887, Nos. 40 and 41.

In looking over my notes, I find I have recorded histories of eighty cases of asthmatics treated during the last five years. Not satisfied with the study of these records, and in order to make my investigation as thorough as possible, and, at the same time, bring the reports up to date, I mailed to each one of these patients, some of whom I had not seen for a considerable time, a printed circular, in which I propounded a series of questions. These questions I will not recapitulate, as they are suggested by the headings in the following analysis of my tables. The last question was, " Please state candidly and without favor what benefit, if any, you have received from the treatment, and to what you attribute your improvement or cure." The following analysis sets forth the result:

Total number of cases of asthma			80
Males	47		
Females	33		
Hay asthma		34	
Males	26		
Females	8		
Perennial asthma		46	
Males	21		
Females	25		

Perennial Asthma—Family History.

Clear in	9
Neurotic	3
Bronchitis and asthma	2
Asthma	4
Asthma and hay fever	4
Bronchitis	2
Asthma, bronchitis and neurosis	1
Phthisis	1
Hay fever	1
Asthma and neurosis	1
Unknown	18
Total	46

Hay Asthma—Family History.

Asthma in	14
Clear in	5
Phthisis and neurosis	1
Neurotic	2
Hay fever	2
Asthma and neurosis	2
" " hay fever	2
" " neurosis and hay fever	1
Neurosis and hay fever	1
Unknown	4
Total	34

The prominent feature shown here is the large preponderance of cases which show a decided neurotic family history; 25 of the 30 cases of hay-asthma being of inherited neurotic habit, when the history is known, while in the perennial form 16 of the 28 cases, in which the history is ascertained, show the neurotic tendency.

Age of first attack.	Perennial asthma.	Hay asthma.
1st ten years of life	5	5
2d " " "	9	7
3d " " "	12	11
4th " " "	6	6
5th " " "	5	4
Over fifty years of age	8	1
From birth	1	...
Total	46	34
Average age of first attack	29 years.	24 years.

Oldest case, 72; youngest, congenital.

We notice that the tables show that the largest number of cases of asthma, in both forms, develop during the third decade of life, while no period is notably exempt. This differs from Salter, who states that most cases develop during the first decade.

Climatic Influence on Hay Asthma.

Greatest relief at high altitude	11
" " " low "	1
" " " sea "	6
" " " New York City	3
Suffer equally everywhere	3
Unknown	10
Total	34

Climatic Influence on Perennial Asthma.

Greatest relief at high altitude	11
Suffering more " "	1
Greatest relief at sea "	2
Suffering more "	7
" equally everywhere	12
Greatest relief at low altitude	1
" " in New York City	1
Unknown	11
Total	46

Combining these two tables in one we find as follows:

Climatic Influences on the Two Forms of Asthma.

Better at high altitudes	22
" at sea shore	8
" in city	9
Unaffected by locality	13
Irregular	9
Effect of locality unknown	19
Total	80

We notice here that whereas in hay fever the seashore affords the greatest relief, after asthmatic symptoms set in the same rule applies to both forms, and that high altitudes are most beneficial; and yet I think no rule can be formulated for the cases as a class. They are essentially fickle, and each one must be advised from personal and individual considerations.

Hay Asthma.

Nasal symptoms immediately preceding attack, such as sneezing with watery discharge from the nose	29
No symptoms preceding attack	5
Total	34

Perennial Asthma.

Nasal symptoms preceding attack	33
No nasal symptoms preceding attack	12
Cutaneous eruption preceding attack	1
Total	46

This showing, it seems to me, is of the greatest importance, as sustaining the original assertion made in the early portion of the paper.

It should be mentioned that many patients entirely ignore the nasal symptoms, in the larger discomfort arising from the dyspnœic attack, and only recall them when their attention is turned in that direction. We see, then, that, of the 80 cases, the asthmatic attack set in with sneezing, etc., in 62.

The one case in which a cutaneous eruption occurred is interesting only with reference to the neurotic explosion.

Hay Asthma.

History of previous catarrhal trouble	23
No history of previous catarrhal trouble	11
Total	34

Perennial Asthma.

History of previous catarrhal trouble	31
No history of previous catarrhal trouble	15
Total	46

We see here that, of the 80 cases, 54 give a history of previous catarrhal trouble. Yet the testimony of patients in this matter is not to be relied upon, as many patients have undoubtedly notable impairment of the nasal respiratory function, without being conscious of suffering from what they call catarrh. Moreover, in a large proportion of nasal disorders, the symptoms are referred, by the individual, to the throat, while "catarrh" is popularly referred to the nose.

Intranasal Condition—Hay Asthma.

Hypertrophic rhinitis	9
" " and deflected septum	12
Polypi and deflected septum	5
Polypi	4
Deflected septum	3
Elongated uvula	1
Total	34

Intranasal Condition—Perennial Asthma.

Hypertrophic rhinitis	13
Nasal polypi	11
Hypertrophic rhinitis and deflected septum	11
Polypi and deflected septum	6
Deflected septum	3
Adenoid and hypertrophic rhinitis	2
Total	46

I have never known a case of hay-fever or asthma to occur in other than an obstructive lesion of the nose or upper air-passages, as will be seen by this table; this was the case in every one of the 80 cases, including the elongated uvula, which became a source of respiratory obstruction.

Treatment—Hay Asthma.

Hypertrophic rhinitis, treatment by caustics: Cured, 7; improved, 6; unimproved, 1.
Deflected septum, operated on by author's nasal saw: Cured, 8; improved, 6.
Nasal polypi treated by snare, without caustics: Cured, 2; improved, 1.
Treatment by snare and septal saw in cases of polypi and deflected septum: Cured, 1; improved, 1.
Cases treated by uvulotomy: Cured, 1.

Treatment—Perennial Asthma.

Hypertrophic rhinitis, treated by caustics: Cured, 8; improved, 5.
Polypi treated by snare, without caustics: Cured, 15; improved, 3.
Deflection of septum operated on by author's nasal saw: Cured, 3; improved, 4; unimproved, 1.
Adenoid growths removed by snare: Cured, 2.

The treatment, as will be seen, has been such as our English friends regard as harsh, and in many cases unwarranted. I think it but justice to say that, in some cases, patients have been unwilling to continue on account of this, and yet, with the use of local anæsthesia, these operations are not painful; it is the nervous strain on this class of patients which has taxed them most severely. That the surgical treatment of nasal diseases is fully justified I think the following table amply demonstrates:

Results of Treatment—Hay Asthma.

Cured	18
Improved	14
Unimproved	1
Unknown	1
Total	34

Results of Treatment—Perennial Asthma.

Cured	28
Improved	12
Unimproved	2
Unknown	4
Total	46

It is not the province of this article to discuss the general therapeutics of asthma, for, in the large majority of these cases, the treatment has been purely local, and yet, in many instances, internal medication has successfully been resorted to, such as the use of the iodides, zinc and belladonna, the three remedies on which I have placed the greatest reliance.

The point to which I desire to give the greatest emphasis is that local treatment of the intranasal disease, which I have invariably found in these cases, affords us by far the most satisfactory method of controlling this distressing and heretofore intractable disease. This seems to be particularly true when the case presents itself for treatment before the age of twenty. In the above tables, four cases of hay-asthma and seven of perennial asthma were under twenty, and all were cured.

It should be remembered that these cases have all come under the observation of a throat specialist, and, hence, a very good reason is immediately apparent why he should not see cases of asthma not dependent on nasal disease. This I have endeavored not to disregard in what has been written, and yet, if the foregoing statements may seem extreme, I can only say that I have endeavored to analyze my cases and report the results with fairness and candor.

PERFORATING ULCERS OF THE FEET, OF AT LEAST TEN YEARS' DURATION, PRECEDING OTHER SYMPTOMS OF TABES DORSALIS:

ASSOCIATED WITH CHARCOT'S JOINT LESION, AND (?) WITH PERFORATING ULCER OF THE TONGUE.

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The following case is interesting on many grounds. It commenced as a typical example of perforating ulcer of the foot; and afterward became symmetrical, affecting the soft parts over the metatarso-phalangeal articulations of both great toes. Sensation was not sufficiently interfered with to prevent pain on walking being his chief complaint. It was the pain that made him beg to have the toe amputated. The ulcers frequently healed after prolonged rest, showing that mechanical injury, in addition to altered innervation, was necessary for the continuance of ulceration. The toes were eventually amputated close to the site of ulceration, and yet the healing of the wounds was fairly rapid and sound. Though now associated with several symptoms of tabes dorsalis, the perforating ulcers existed for six or eight years, at least, without any discoverable sign of locomotor ataxia, and even now the *ataxic* symptoms proper are very slight indeed.

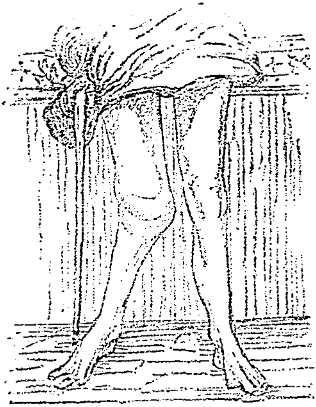
If we accept the positive assurance of the patient that the ulcer of the tongue was not preceded by any stage of induration, we must receive it as an example of a very rare condition: namely, perforating ulcer of the tongue; and, moreover, a perforating ulcer which has healed.

On the whole, it appears to be a case of tabes dorsalis in which the peripheral multiple neuritis, which is almost invariably found at some stage, has appeared early, as shown by the ptosis, optic atrophy, joint lesion (knee) and perforating ulcers. It is doubtful whether it may not be an instance of true tabes dorsalis commencing in a peripheral neuritis, as suggested recently by Professor Leyden, and affecting the posterior columns of the cord secondarily. In this suggestion I have assumed peripheral neuritis to be the most probable cause of the joint lesion as well as of the perforating ulcers. In this connection the "Report of the Committee on Joint Disease in Connection with Locomotor Ataxia," published in vol. xx. of the *Transactions of the Clinical Society of London*, is interesting, and especially the case of J. Griffiths related on page 299.

CASE.—W. W., aged forty-one, a bricklayer's laborer, and formerly a laborer at the Gas Works, was admitted into the Nottingham General Hospital, under my care, on March 10, 1887, complaining of swelling of the left knee.

He had gonorrhœa and a sore on the penis twenty years ago, but does not remember having any rash or sore throat. We may take it then, that, if he had syphilis, the secondary symptoms were mild. He has had no tertiary affection, unless the condition of the tongue to be presently described was such. About fifteen years ago, a railway metal crushed his right great toe, and burst the skin. The wound soon healed, and remained sound for four or five years. After that, a sore appeared on the plantar surface of the right great toe opposite the metatarso-phalangeal articulation. In a few months, a similar sore appeared in exactly the same position on the left great toe. With rest these sores readily healed, but as readily broke out again as soon as he began to walk and work. I first saw him in 1879, when he was in the hospital under the care of my colleague, Mr. Wright. The ulcers were rounded and deep, and a sinus led down to the sesamoid bones which necrosed and eventually were exfoliated. The ulcers were surrounded by a zone of very greatly indurated and thickened epithelium. The patient was anxious to have his toes amputated, but eventually the ulcers healed, as they had done before. At this time there were no symptoms of tabes. After many vicissitudes, he determined to have the left toe off. It was amputated about four years ago, and the wound healed well. For some months after that he was able to work as a bricklayer's laborer, and go up a ladder with a load of bricks. There was evidently no ataxia at this period. Two years later the right great toe was similarly removed at his urgent request, by my colleague, Mr. A. C. Taylor, on account of the pain of the ulcer on its under surface. I have no record as to the existence or not of any area of anæsthesia, or even of diminished sensibility, but there is no doubt that on both feet the ulcers were painful during walking, and it was because of this pain that he insisted on having the toes removed. It is curious that a neuritis sufficient to affect gravely the nutrition of the part should not have interfered more with sensation. Since he lost both toes he has not been able to work.

FIG. 1.



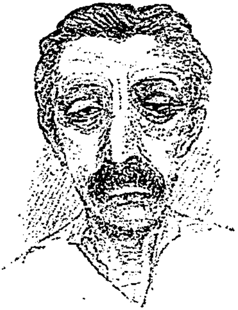
Charcot's joint lesion affecting the right knee-joint. (From a photograph.) The loss of both great toes is shown.

Eight or nine weeks before his admission into the hospital on the present occasion, his right leg became swollen from the knee to the ankle, and later the swelling extended up the thigh. It was not due to any injury, so far as he is aware. There was much pain in the knee at first. Now the swelling of the leg and thigh is gone, as is also the pain; but the knee remains much enlarged (Fig. 1). There is no distinct fluctuation, but much pulpy swelling, and considerable enlargement of the ends of the bones. The internal lateral ligaments have yielded, and there is considerable genu valgum on standing, which he can do without pain. The movements of flexion and extension are free, and he can walk with the aid of a stick.

There is a large depression about the middle of the left border of the tongue. It is quite soft, smooth and soundly healed. He thinks he bit his tongue about a year ago, and that an ulcer followed. He is sure it was not preceded by a hard lump. It is doubtful whether this has been a gumma or a perforating ulcer. The history and the absence of induration and puckering suggest the latter.

He has some cardiac irregularity on excitement, but no murmur. He sleeps moderately well, his speech is very thick, and he complains of occasional shooting pains. His sight is fairly good. He has a very morose expression, with thick, heavy lips and moderate double ptosis (Fig. 2). There is commencing double optic atrophy. His hearing is good, but taste is somewhat defective, though he can distinguish salt and sugar. No affection of the muscular sense could be detected. There is complete absence of knee-jerk on the left, but on the right the knee is too much swollen for any definite result to be obtained. He cannot stand with the eyes closed. He frequently has some difficulty in passing water, and occasionally passes it involuntarily. Common sensation appears to be blunted, but nowhere lost. With the eyes blindfolded he could generally tell when he was touched on the leg with a feather, and usually distinguished the proper side, *i. e.*, right or left; but he was very frequently wrong in localizing. Occasionally, after he had been touched many times, he quite ceased to be able to tell correctly the spot touched; and even continued to feel touches and localize them for a minute or two after the experiments had ceased. He could localize correctly a touch with the finger at first, and distinguish it from a knife-point, but afterward he confused the two. There was no very evident delay in the perception of sensations. The sense of contact is less acute on the stump of the right toe than on that of the left. Heat and cold can only be distinguished in the legs when the water in the test-tube is boiling.

FIG. 2.

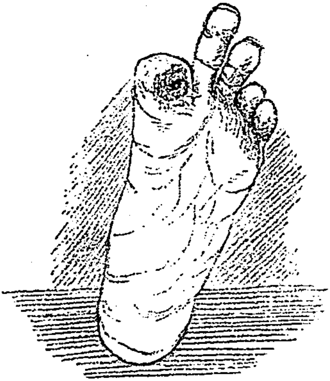


From a photograph; showing the double ptosis, thick lips and general moroso expression.

He can place the legs in any position indicated, and also tell in what position they have been placed for him. In the upper extremities common sensation is acute as compared with the lower. Sensation to pain is moderately good except on the left forefinger, where a pin stuck into the skin, so as to stand up, was said to be a touch with a finger.

He remained in the hospital several months, but the knee did not improve much and he went out. After he had begun to go about again with the aid of a stick, a fresh typical perforating ulcer, surrounded by a zone of thickened epithelium, developed on the under surface of the stump of the left great toe (Fig. 3). In a month or two, however, it was healed.

Fig. 3.



Perforating ulcer on the stump of the left great toe which had been amputated four years previously for a perforating ulcer higher up.

A year later, in March, 1888, he remained in very much the same condition. The ulcer remains healed, but he has had a fresh one on the upper surface of the stump of the left great toe. This was evidently caused by the friction of his slipper and eventually healed. He only walks about the house with a stick. The knee remains unaltered, except that it gives way more on walking, but it continues painless. The tongue has not broken out afresh. He walks without any incoördination of movement; there is no jerking of the limbs or stamping of the heels.